

**WEST**[Help](#)[Logout](#)[Interrupt](#)[Main Menu](#)[Search Form](#)[Posting Counts](#)[Show S Numbers](#)[Edit S Numbers](#)[Preferences](#)[Cases](#)**Search Results -**

Terms	Documents
(galactose-beta 1,3-N-acetylgalactosamine-alpha-R) or (N-acetylglucosamine-beta 1,3-N-acetylgalactosamine-alpha-R) or (beta 1,6-N-acetylglucosaminyltransferase) or (C2/4GnT)	5

**Database:**

US Patents Full-Text Database	▲
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**Search:**

L2

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side by side**Hit Count   Set Name**  
result set

DB=USPT,PGPB,JPAB,EPAB,DWPI; PLUR=YES; OP=ADJ

<u>L2</u>	(galactose-beta 1,3-N-acetylgalactosamine-alpha-R) or (N-acetylglucosamine-beta 1,3-N-acetylgalactosamine-alpha-R) or (beta 1,6-N-acetylglucosaminyltransferase) or (C2/4GnT)
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5   L2

<u>L1</u>	PCT/DK99/00677
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1   L1

END OF SEARCH HISTORY

**WEST**☐  

L2: Entry 1 of 5

File: PGPB

Oct 17, 2002

DOCUMENT-IDENTIFIER: US 20020150968 A1

TITLE: Glycoconjugate and sugar nucleotide synthesis using solid supports

Detail Description Paragraph (116):

[0128] The inventors also recognize that organisms that naturally express one or more enzymes, or have been engineered to express one or more enzymes, required for a particular glycoconjugate synthesis scheme may be useful. Examples include *Escherichia coli* which expresses the ceramide glucosyltransferase gene derived from human melanoma cell line SK-Mel-28 (Proc. Natl. Acad. Sci. USA, 1996, 93:4638), human melanoma cell line WM266-4 which produces beta 1,3-galactosyltransferase (ATCC CRL 1676), recombinant cell line such as namalwa cell line KJM-1 or the like which contains the beta 1,3-galactosyltransferase gene derived from the human melanoma cell line WM266-4 (Japanese Published Unexamined Patent Application No. 181759/94), *Escherichia coli* (EMBO J., 1990, 9, 3171) or *Saccharomyces cerevisiae* (Biochem. Biophys. Res. Commun., 1994, 201, 160) which expresses the beta 1,4-galactosyltransferase gene derived from human HeLa cells, COS-7 cell line (ATCC CRL 1651) which expresses the rat beta 1,6-N-acetylglucosaminyltransferase gene (J. Biol. Chem., 1993, 268: 15381), Sf9 cell line which expresses human N-acetylglucosaminyltransferase gene (J. Biochem., 1995, 118: 568), *Escherichia coli* which expresses human glucuronosyltransferase (Biochem. Biophys. Res. Commun., 1993, 196: 473), namalwa cell line which expresses human alpha 1,3-fucosyltransferase (J. Biol. Chem., 1994, 269: 14730), COS-1 cell line which expresses human alpha 1,3/1,4-fucosyltransferase (Genes Dev., 1990, 4: 1288), COS-1 cell line which expresses human alpha 1,2-fucosyltransferase (Proc. Natl. Acad. Sci. USA., 1990, 87: 6674), COS-7 cell line which expresses chicken alpha 2,6-sialyltransferase (Eur. J. Biochem., 1994, 219: 375), COS cell line which expresses human alpha 2,8-sialyltransferase (Proc. Natl. Acad. Sci. USA., 1994, 91: 7952), *Escherichia coli* which expresses beta 1,3-N-acetylglucosaminyltransferase-, beta 1,4-galactosyltransferase, beta 1,3-N-acetylgalactosaminyltransferase or alpha 1,4-galactosyltransferase derived from *Neisseria* (WO 96/10086), *Escherichia coli* which expresses *Neisseria*-derived alpha 2,3-sialyltransferase (J. Biol. Chem., 1996, 271: 28271), *Escherichia coli* which expresses *Helicobacter pylori*-derived alpha 1,3-fucosyltransferase (J. Biol. Chem., 1997, 272: 21349 and 21357), and *Escherichia coli* which expresses yeast-derived alpha 1,2-mannosyltransferase (J. Org. Chem., 1993, 58: 3985). Such organism when further complemented with additional sugar-nucleotide regenerating enzymes will be useful in the methods of the present invention.

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L2: Entry 3 of 5

File: EPAB

Mar 16, 1995

DOCUMENT-IDENTIFIER: WO 9507020 A1

TITLE: EXPRESSION OF THE DEVELOPMENTAL I ANTIGEN BY A CLONED HUMAN cDNA ENCODING A BETA-1,6-N-ACETYLGLUCOSAMINYLTRANSFERASE

Abstract (1):

The present invention provides an isolated nucleic acid molecule encoding both a soluble and membrane-bound human beta -1,6-N-acetylglucosaminyltransferase, the I-branching enzyme (IGnT). The invention also provides vectors containing the isolated nucleic acid molecule encoding human IGnT as well as recombinant host cells transformed with the vectors. The invention further provides a method of preparing a membrane-bound form of human IGnT and methods of preparing and purifying soluble human IGnT and active fragments of either form. Also provided are antisense oligonucleotides complementary to a nucleic acid molecule encoding a human IGnT or an active fragment thereof, antibodies directed to the human IGnT, pharmaceutical compositions related to the human IGnT and transgenic nonhuman mammals expressing DNA encoding normal or mutant human IGnT. Also provided are methods for regulating the expression of human IGnT and methods for modifying a biological function mediated by the regulatory activity of human IGnT. Methods of detecting the presence of linear polylactosaminoglycans expressing i antigenic determinants on a cell surface also are provided.

**WEST**

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L2: Entry 4 of 5

File: DWPI

Jun 27, 2002

DERWENT-ACC-NO: 2000-423407  
DERWENT-WEEK: 200245  
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TITLE: New nucleic acid molecule encoding UDP-N-acetylglucosamine useful as probe for the detection of specified glucoseaminyltransferase from other species and related organisms

Basic Abstract Text (1):

NOVELTY - An isolated nucleic acid molecule (I), encoding UDP-N-acetylglucosamine: galactose- beta 1,3-N-acetylgalactosamine- alpha -R/N-acetylglucosamine- beta 1,3-N-acetylgalactosamine- alpha -R beta 1,6-N-acetylglucosaminyltransferase (C2/4GnT) or a fragment, is new.

Basic Abstract Text (7):

(5) method of producing C2/4GnT polypeptide; and

Basic Abstract Text (8):

(6) method for identification of variations in beta C2/4GnT gene comprises amplifying C2/4GnT genomic regions by PCR and detecting the presence of sequence variation by DNA sequencing, single-strand conformational polymorphism (SSCP) or mismatch mutation.

Basic Abstract Text (9):

USE - (I) is useful as a probe for the detection of C2/4GnT from other species and related organisms and for the recombinant production of C2/4GnT polypeptide.

Equivalent Abstract Text (1):

NOVELTY - An isolated nucleic acid molecule (I), encoding UDP-N-acetylglucosamine: galactose- beta 1,3-N-acetylgalactosamine- alpha -R/N-acetylglucosamine- beta 1,3-N-acetylgalactosamine- alpha -R beta 1,6-N-acetylglucosaminyltransferase (C2/4GnT) or a fragment, is new.

Equivalent Abstract Text (7):

(5) method of producing C2/4GnT polypeptide; and

Equivalent Abstract Text (8):

(6) method for identification of variations in beta C2/4GnT gene comprises amplifying C2/4GnT genomic regions by PCR and detecting the presence of sequence variation by DNA sequencing, single-strand conformational polymorphism (SSCP) or mismatch mutation.

Equivalent Abstract Text (9):

USE - (I) is useful as a probe for the detection of C2/4GnT from other species and related organisms and for the recombinant production of C2/4GnT polypeptide.

**WEST****End of Result Set**☐

Generate Collection

Print

L2: Entry 5 of 5

File: DWPI

Jun 16, 1998

DERWENT-ACC-NO: 1998-361697

DERWENT-WEEK: 199831

COPYRIGHT 2002 DERWENT INFORMATION LTD

TITLE: New nucleic acid sequences and their complementary sequences - useful for producing fragment of recombinant human I-branching -1,6-N-acetyl-gluc osaminyln-transferase polypeptide

**Basic Abstract Text (1):**

Nucleic acid selected from: (a) a nucleotide sequence (I), which is linear and has 378 base pairs, as given in the specification; (b) a nucleotide sequence (II) complementary to (I); and (c) a mixture of (I) and (II) comprising single or double stranded nucleic acid molecule. Also claimed are: (A) a nucleic acid selected from a nucleotide sequence (III), which is linear and has 1807 base pairs, as given in the specification; a nucleotide sequence (IV) complementary to (III); and a mixture of (III) and (IV) comprising single or double stranded nucleic acid molecule; (B) a nucleic acid selected from a nucleotide sequence (V), which is linear and has 126 base pairs, as given in the specification; a nucleotide sequence (VI) complementary to (V); and a mixture of (V) and (VI) comprising single or double stranded nucleic acid molecule; (C) a nucleic acid selected from a nucleotide sequence (VII), which is linear and has 400 base pairs, as given in the specification; a nucleotide sequence (VIII) complementary to (VII); and a mixture of (VII) and (VIII) comprising single or double stranded nucleic acid molecule; (D) a vector (IX) containing a nucleic acid molecule comprising a nucleotide sequence selected from (I), (III), (V) and (VII); (E) a host cell containing (IX); (F) a process for obtaining a purified soluble active fragment of recombinant human I-branching beta-1,6-N-acetylglucosaminylntransferase polypeptide (IGnT), comprising: (i) introducing a nucleic acid molecule (selected from (I) or (III)) encoding the soluble active fragment of human IGnT into a host cell; (ii) expressing the soluble active fragment of human IGnT; and (iii) purifying the expressed soluble active fragment of IGnT; and (G) a process for obtaining a host cell expressing a recombinant human IGnT, or a soluble active fragment of it, comprising: (i') introducing a nucleic acid sequence selected from (I), (III), (V) and (VII), into a host cell; and (ii') expressing the recombinant human IGnT or fragment of it in the cell.

**WEST**[Generate Collection](#)[Print](#)**Search Results - Record(s) 1 through 5 of 5 returned.**☐ 1. Document ID: US 20020150968 A1

L2: Entry 1 of 5

File: PGPB

Oct 17, 2002

PGPUB-DOCUMENT-NUMBER: 20020150968  
PGPUB-FILING-TYPE: new  
DOCUMENT-IDENTIFIER: US 20020150968 A1

TITLE: Glycoconjugate and sugar nucleotide synthesis using solid supports

PUBLICATION-DATE: October 17, 2002

## INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Wang, Peng G.	Troy	MI	US	
Chen, Xi	Norristown	PA	US	

US-CL-CURRENT: [435/53](#); [435/175](#), [435/68.1](#), [435/96](#)

<a href="#">Full</a>	<a href="#">Title</a>	<a href="#">Citation</a>	<a href="#">Front</a>	<a href="#">Review</a>	<a href="#">Classification</a>	<a href="#">Date</a>	<a href="#">Reference</a>	<a href="#">Sequences</a>	<a href="#">Attachments</a>	<a href="#">Claims</a>	<a href="#">KMC</a>	<a href="#">Draw Desc</a>	<a href="#">Image</a>
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☐ 2. Document ID: US 20020081656 A1

L2: Entry 2 of 5

File: PGPB

Jun 27, 2002

PGPUB-DOCUMENT-NUMBER: 20020081656  
PGPUB-FILING-TYPE: new  
DOCUMENT-IDENTIFIER: US 20020081656 A1

TITLE: UDP-N-acetylglucosamine: Galactose-beta1,3-N-acetylgalactosamine-alpha-R /  
N-acetylglucosamine-beta1,3,-N-acetylgalactosamine-alpha-R (GlcNAc to GalNAc)  
beta1,6-N-acetylglucosaminyltransferase, [C2/4GnT](#)

PUBLICATION-DATE: June 27, 2002

## INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Clausen, Henrik	Holte		DK	
Schwientek, Tilo	Bronshoj		DK	

US-CL-CURRENT: [435/69.1](#); [435/193](#), [435/320.1](#), [435/325](#), [435/6](#), [536/23.2](#)

<a href="#">Full</a>	<a href="#">Title</a>	<a href="#">Citation</a>	<a href="#">Front</a>	<a href="#">Review</a>	<a href="#">Classification</a>	<a href="#">Date</a>	<a href="#">Reference</a>	<a href="#">Sequences</a>	<a href="#">Attachments</a>	<a href="#">Claims</a>	<a href="#">KMC</a>	<a href="#">Draw Desc</a>	<a href="#">Image</a>
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☐ 3. Document ID: WO 9507020 A1

L2: Entry 3 of 5

File: EPAB

Mar 16, 1995

PUB-NO: WO009507020A1

DOCUMENT-IDENTIFIER: WO 9507020 A1

TITLE: EXPRESSION OF THE DEVELOPMENTAL I ANTIGEN BY A CLONED HUMAN cDNA ENCODING A BETA-1,6-N-ACETYLGLUCOSAMINYLTRANSFERASE

Full	Title	Citation	Front	Review	Classification	Date	Reference	Sequences	Attachments	Claims	KWIC	Draw Desc	Image
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☒ 4. Document ID: US 20020081656 A1 WO 200034449 A2 AU 200015037 A EP 1135472 A2

L2: Entry 4 of 5

File: DWPI

Jun 27, 2002

DERWENT-ACC-NO: 2000-423407

DERWENT-WEEK: 200245

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TITLE: New nucleic acid molecule encoding UDP-N-acetylglucosamine useful as probe for the detection of specified glucoseaminyltransferase from other species and related organisms

Full	Title	Citation	Front	Review	Classification	Date	Reference	Sequences	Attachments	Claims	KWIC	Draw Desc	Image
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☐ 5. Document ID: US 5766910 A

L2: Entry 5 of 5

File: DWPI

Jun 16, 1998

DERWENT-ACC-NO: 1998-361697

DERWENT-WEEK: 199831

COPYRIGHT 2002 DERWENT INFORMATION LTD

TITLE: New nucleic acid sequences and their complementary sequences - useful for producing fragment of recombinant human I-branching -1,6-N-acetyl-gluc osaminyln-transferase polypeptide

Full	Title	Citation	Front	Review	Classification	Date	Reference	Sequences	Attachments	Claims	KWIC	Draw Desc	Image
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Terms	Documents
(galactose-beta 1,3-N-acetylgalactosamine-alpha-R) or (N-acetylglucosamine-beta 1,3-N-acetylgalactosamine-alpha-R) or (beta 1,6-N-acetylglucosaminyltransferase) or (C2/4GnT)	5

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INDEX 'ADISALERTS, ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, AQUASCI,  
BIOBUSINESS, BIOCOMMERCE, BIOSIS, BIOTECHABS, BIOTECHDS, BIOTECHNO,  
CABA,  
CANCERLIT, CAPLUS, CEABA-VTB, CEN, CIN, CONFSCI, CROPB, CROPU, DDFB,  
DDFU, DGENE, DRUGB, DRUGLAUNCH, DRUGMONOG2, ...' ENTERED AT 12:19:40 ON  
18 OCT 2002

SEA (GALACTOSE-BETA 1,3-N-ACETYLGALACTOSAMINE-ALPHA-R) OR  
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0\* FILE ADISNEWS  
1\* FILE AGRICOLA  
3\* FILE ANABSTR  
0\* FILE AQUASCI  
2\* FILE BIOBUSINESS  
0\* FILE BIOCOMMERCE  
99\* FILE BIOSIS  
0\* FILE BIOTECHABS  
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0\* FILE DRUGMONOG2  
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L1 QUE (GALACTOSE-BETA 1,3-N-ACETYLGALACTOSAMINE-ALPHA-R) OR  
 (N-AC

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FILE 'MEDLINE, CAPLUS, SCISEARCH, BIOSIS, EMBASE, BIOTECHNO, CANCERLIT, ESBIODBASE' ENTERED AT 12:27:52 ON 18 OCT 2002

L2 182 S L1 AND (CDNA OR CLONE)  
 L3 65 DUP REM L2 (117 DUPLICATES REMOVED)  
 L4 0 S L3 AND (C2!4GNT)  
 L5 49 S L3 AND HUMAN  
 L6 16 S L3 AND (C2 OR GNT)

L6 ANSWER 1 OF 16 MEDLINE  
 ACCESSION NUMBER: 2001420800 MEDLINE  
 DOCUMENT NUMBER: 21362968 PubMed ID: 11469797  
 TITLE: The widespread effect of beta 1,4-galactosyltransferase on N-glycan processing.  
 AUTHOR: Fukuta K; Abe R; Yokomatsu T; Minowa M T; Takeuchi M; Asanagi M; Makino T  
 CORPORATE SOURCE: Life Science Laboratory, Mitsui Chemicals, Incorporated, 1144 Togo, Mobara, Chiba 297-0017, Japan.. kazuhiro.fukuta@mitsui-chem.co.jp  
 SOURCE: ARCHIVES OF BIOCHEMISTRY AND BIOPHYSICS, (2001 Aug 1) 392 (1) 79-86.  
 Journal code: 0372430. ISSN: 0003-9861.  
 PUB. COUNTRY: United States  
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
 LANGUAGE: English  
 FILE SEGMENT: Priority Journals  
 ENTRY MONTH: 200109  
 ENTRY DATE: Entered STN: 20010910  
 Last Updated on STN: 20010910  
 Entered Medline: 20010906

AB We investigated beta 1,4-GalT (UDP-galactose: beta-d-N-acetylglucosaminide beta 1,4-galactosyltransferase) in terms of intracellular competition with **GnT-IV** (UDP-N-acetylglucosamine: alpha1,3-d-mannoside beta1,4-N-acetylglucosaminyltransferase) and **GnT-V** (UDP-N-acetylglucosamine: alpha1,6-d-mannoside **beta 1, 6-N-acetylglucosaminyltransferase**). The beta 1,4-GalT-I gene was introduced into Chinese hamster ovary (CHO) cells producing human interferon (hIFN)-gamma (IM4/V/IV cells) and five **clones** expressing various levels of beta 1,4-GalT were isolated. As we previously reported, parental IM4/V/IV cells express high levels of **GnT-IVa** and -V and produce hIFN-gamma having primarily tetraantennary sugar chains. The branching of sugar chains on hIFN-gamma was suppressed in the beta 1,4-GalT-enhanced **clones** to a level corresponding to the intracellular activity of beta 1,4-GalT relative to **GnTs**. Moreover, the contents of hybrid-type and high-mannose-type sugar chains increased in these **clones**. The results showed that beta 1,4-GalT widely affects N-glycan processing by competing with **GnT-IV**, **GnT-V**, and alpha-mannosidase II in cells and also by some other mechanisms that suppress the conversion of high-mannose-type sugar chains to the hybrid type.  
 Copyright 2001 Academic Press.

L6 ANSWER 2 OF 16 MEDLINE  
 ACCESSION NUMBER: 2001160699 MEDLINE  
 DOCUMENT NUMBER: 21158291 PubMed ID: 11261840  
 TITLE: Modulation of the basal activity of phosphatidylinositol-3-kinase/protein kinase B signaling pathway in human hepatocarcinoma cells.  
 AUTHOR: Guo H B; Shen Z H; Huang C X; Ma J; Huang Y; Chen H L  
 CORPORATE SOURCE: Key Laboratory of Glycoconjugate Research, Ministry of Health, Shanghai Medical University, China.  
 SOURCE: GLYCOCONJUGATE JOURNAL, (2000 May) 17 (5) 315-22.  
 Journal code: 8603310. ISSN: 0282-0080.  
 PUB. COUNTRY: United States  
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English  
FILE SEGMENT: Priority Journals  
ENTRY MONTH: 200108  
ENTRY DATE: Entered STN: 20010820  
Last Updated on STN: 20020420  
Entered Medline: 20010816

AB The modulation of **GnT**-V activity by signaling molecules in PI-3-K/PKB pathway in human hepatocarcinoma cell line 7721 was studied. **GnT**-V activity was determined after the transfection of sense or antisense **cDNA** of PKB into the cells, as well as the addition of activators, specific inhibitors, and the antibodies to the enzyme assay system or culture medium. It was found that the basal activity of **GnT**-V was up regulated by the sense and down regulated by the antisense **cDNA** of PKB transfected into 7721 cells. **GnT**-V was activated by PIP2, PIP3 or GTPgamma[S] added to the assay system, and the activation of PIP2 or GTPgamma[S] was abolished by LY2940002, a specific inhibitor of PI-3-K, but the activation of PIP3 was not attenuated by LY2940002. In addition, **GnT**-V activity in cultured parental or H-ras transfected cells was inhibited by the antibody against PKB or PI-3-K. These findings demonstrated the involvement of PI-3-K/PKB signaling pathway in the regulation of **GnT**-V. Moreover, ET18-OCH3, an inhibitor of Raf translocation and PI-PLC enzyme, which produces the activator of PKC, as well as the antibodies against Raf-1 or MEK also inhibited **GnT**-V activity in the parental and H-ras transfected cells. The inhibitory rates, however, were less in the transfected cells than those in the parental cells. These results reveal that in parental and H-ras transfected 7721 cells, the basal activity of **GnT**-V is also regulated by the Ras/Raf-1/MEK/MAPK cascade in addition to PI-3-K/PKB signaling pathway. The significance of these two pathways in the regulation of **GnT**-V and their relations to the activation of PKC previously reported by our laboratory (Ju TZ et al., 1995 Glyconjugate J 12, 767-772) was discussed.

L6 ANSWER 3 OF 16 MEDLINE  
ACCESSION NUMBER: 2001082782 MEDLINE  
DOCUMENT NUMBER: 20428761 PubMed ID: 10972975  
TITLE: Down-regulation of N-acetylglucosaminyltransferase V by tumorigenesis- or metastasis-suppressor gene and its relation to metastatic potential of human hepatocarcinoma cells.  
AUTHOR: Guo H B; Liu F; Zhao J H; Chen H L  
CORPORATE SOURCE: Key Laboratory of Glycoconjugate Research, Ministry of Health, Department of Biochemistry, Shanghai Medical University, Shanghai, People's Republic of China.  
SOURCE: JOURNAL OF CELLULAR BIOCHEMISTRY, (2000 Sep 7) 79 (3) 370-85.  
Journal code: 8205768. ISSN: 0730-2312.  
PUB. COUNTRY: United States  
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
LANGUAGE: English  
FILE SEGMENT: Priority Journals  
ENTRY MONTH: 200101  
ENTRY DATE: Entered STN: 20010322  
Last Updated on STN: 20010322  
Entered Medline: 20010105

AB The effects of transfection of the metastasis suppressor gene nm23-H1 and cell-cycle related tumor-suppressor gene p16 on the activity of N-acetylglucosaminyltransferase V (**GnT**-V) and their relations to cancer metastatic potential were investigated. After transfection of nm23-H1 into 7721 human hepatocarcinoma cells and A549 human lung cancer cells, the activities of **GnT**-V were decreased by 28%-42% in the cells. In contrast, when p16 was transfected into these two cell lines, the decrease of **GnT**-V activity was only observed in A549 cells. This was probably to be due to the obvious expression of p16 gene in parental 7721 cells and the deletion of p16 in A549 cells. The decrease

of

GnT-V mRNA was only observed in nm23-H1-transfected cells, but not in p16-transfected A549 cells, suggesting that the two genes regulated GnT-V via different mechanisms. Horseradish peroxidase (HRP)-lectin staining showed that the 7721 cells transfected with nm23-H1 or the A549 cells transfected with p16 displayed a decreased intensity with HRP-leucoagglutinating phytohemagglutinin and increased intensity with HRP-concanavalin A, indicating the decline of beta1,6 N-acetylglucosamine branching structure on the asparagine-linked glycans of cell-surface and intracellular glycoproteins. The nm23-H1 transfected 7721 cells also displayed some changes in metastasis-related phenotypes, including the increase in cell adhesion to fibronectin (Fn), the decline in cell adhesion to laminin (Ln), and the decreased cell migration and invasion through matrigel. Transfection of antisense GnT-V cDNA into 7721 cells resulted in a decrease of GnT-V activity, an increase of cell adhesion to Fn or Ln, and a decrease in cell migration and invasion through matrigel. These phenotypes bore similarity to those of the 7721 cells transfected with nm23-H1. Our findings indicate that the down-regulation of GnT-V by nm23-H1 contributes to the alterations in metastasis-related phenotypes, and is an important molecular mechanism of metastasis suppression mediated by nm23-H1.

L6 ANSWER 4 OF 16 MEDLINE  
 ACCESSION NUMBER: 2000001950 MEDLINE  
 DOCUMENT NUMBER: 20001950 PubMed ID: 10529394  
 TITLE: Increased susceptibility to apoptosis of human hepatocarcinoma cells transfected with antisense N-acetylglucosaminyltransferase V cDNA.  
 COMMENT: Erratum in: Biochem Biophys Res Commun 1999 Dec 20;266(2):615  
 AUTHOR: Guo H B; Liu F; Chen H L  
 CORPORATE SOURCE: Ministry of Health, Shanghai Medical University, Shanghai, 200032, China.  
 SOURCE: BIOCHEMICAL AND BIOPHYSICAL RESEARCH COMMUNICATIONS, (1999 Oct 22) 264 (2) 509-17.  
 Journal code: 0372516. ISSN: 0006-291X.  
 PUB. COUNTRY: United States  
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
 LANGUAGE: English  
 FILE SEGMENT: Priority Journals  
 ENTRY MONTH: 199912  
 ENTRY DATE: Entered STN: 20000113  
 Last Updated on STN: 20000314  
 Entered Medline: 19991207

AB The antisense cDNA of N-acetylglucosaminyltransferase V (GnT-V, EC 2. 4.1.155) was constructed as pCDNA3/GnT-V-AS plasmid and transfected into 7721 cells, a human hepatocarcinoma cell line. The transfection was confirmed with Northern blot. By using HPLC and HRP-lectin staining, it was found that the cells transfected with pCDNA3/GnT-V-AS (GnT-V-AS/7721) expressed less GnT-V activity and beta-1,6-GlcNAc branching in the cell glycoproteins compared with the cells mock-transfected with the vector pCDNA3 (pCDNA3/7721). The growth rate of GnT-V-AS/7721 was decreased in serum-containing medium, while the cell death was accelerated in serum-free medium. The GnT-V-AS/7721 cells were more susceptible to the apoptosis induced by ATRA than the mock-transfected cells. This was evidenced by the obvious appearance of a hypoploid sub-G(1) fraction in the DNA histogram using FCM analysis, the more condensed new moon-type nuclei under morphological observation, and the more intensive TUNEL reaction for assaying the fragmented DNA. At the same time as GnT-V down-regulation by GnT-V-AS, an increase of another N-acetylglucosaminyltransferase,

GnT-III (EC 2.4.1.144), was observed, and the biological significance of this finding was discussed.  
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L6 ANSWER 5 OF 16 MEDLINE  
ACCESSION NUMBER: 1999367413 MEDLINE  
DOCUMENT NUMBER: 99367413 PubMed ID: 10438459  
TITLE: Regulation of the GnT-V promoter by transcription factor Ets-1 in various cancer cell lines.  
AUTHOR: Ko J H; Miyoshi E; Noda K; Ekuni A; Kang R; Ikeda Y; Taniguchi N  
CORPORATE SOURCE: Department of Biochemistry, Osaka University Medical School, Room B1, 2-2 Yamadaoka, Suita, Osaka 565-0871, Japan.  
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (1999 Aug 13) 274 (33) 22941-8.  
Journal code: 2985121R. ISSN: 0021-9258.  
PUB. COUNTRY: United States  
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
LANGUAGE: English  
FILE SEGMENT: Priority Journals  
ENTRY MONTH: 199909  
ENTRY DATE: Entered STN: 19990913  
Last Updated on STN: 19990913  
Entered Medline: 19990901

AB Although the precise role of oligosaccharides in metastasis is presently unknown, numerous studies suggest that the beta1-6 branching structure of N-linked oligosaccharides plays a role in tumor metastasis. N-Acetylglucosaminyltransferase V (GnT-V), which catalyzes the formation of the beta1-6 branch, therefore appears to play a crucial role in tumor metastasis. Recently, we demonstrated that the expression of the GnT-V gene is regulated by a transcriptional factor, Ets-1 (Kang, R., Saito, H., Ihara, Y., Miyoshi, E., Koyama, N., Sheng, Y., and Taniguchi, N. (1996) J. Biol. Chem. 271, 26706-26712). In this study, we report an investigation of the general requirement for Ets-1 in the expression of GnT-V in cancer cell lines. In 16 cancer cell lines, the levels of GnT-V mRNA were closely correlated with ets-1 expression ( $r = 0.97$ ;  $p < 0.0001$ ). An increase in ets-1 levels by transfection of its cDNA led to an enhancement in GnT-V expression in cells that normally expressed low levels of ets-1. In contrast, the transfection of dominant negative ets-1 into cells that express high levels of ets-1 resulted in a decrease in GnT-V expression. Although Ets-1 cooperates with c-Jun in certain gene expressions, this was not the case in the regulation of the GnT-V gene. These results suggest that Ets-1 plays a significant role in regulating the expression of GnT-V in a variety of cancers and might be involved in the potential for malignancy via the action of GnT-V.

L6 ANSWER 6 OF 16 MEDLINE  
ACCESSION NUMBER: 1998001705 MEDLINE  
DOCUMENT NUMBER: 98001705 PubMed ID: 9341170  
TITLE: Tissue-specific regulation of mouse core 2 beta-1,6-N-acetylglucosaminyltransferase.  
AUTHOR: Sekine M; Nara K; Suzuki A  
CORPORATE SOURCE: Department of Membrane Biochemistry, Tokyo Metropolitan Institute of Medical Science, Bunkyo-ku, Tokyo 113, Japan.  
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (1997 Oct 24) 272 (43) 27246-52.  
Journal code: 2985121R. ISSN: 0021-9258.  
PUB. COUNTRY: United States  
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
LANGUAGE: English  
FILE SEGMENT: Priority Journals

OTHER SOURCE: GENBANK-D87332; GENBANK-D87333  
ENTRY MONTH: 199601  
ENTRY DATE: Entered STN: 19980109  
Last Updated on STN: 20010813  
Entered Medline: 19971128

AB Mouse kidney beta-1,6-GlcNAc-transferase (**GNT**) is the key enzyme for the synthesis of a glycosphingolipid (Galbeta1-4(Fucalpha1-3)GlcNAcbeta1-6(Galbeta1-3)GalNAcbeta1-3Galalpha1-4Galbeta1-4Glcbeta1-ceramide) that contains the LeX trisaccharide epitope at its nonreducing terminus. The expression of this glycolipid in the kidney is polymorphic; it is expressed in BALB/c but not DBA/2 mice; and a single autosomal gene (*Gsl5*) is responsible for this polymorphism. We report here the **cDNA** sequence that encodes the kidney **GNT** of BALB/c mice, which possess a wild-type *Gsl5* gene. The deduced amino acid sequence

exhibits 84% identity to that of human core 2 beta-1,6-GlcNAc-transferase, which suggests that kidney **GNT** is a mouse homologue of human core 2 beta-1, 6-GlcNAc-transferase. The **GNT** mRNA is expressed abundantly in the kidney, but was not detected in other BALB/c organs or in the kidneys of DBA/2 mice by Northern blot analysis. In addition, we were able to **clone** and sequence another homologous **cDNA** from the submandibular gland. The two sequences differ only in their 5'-untranslated region. The submandibular gland type of **cDNA** was detected in various organs of DBA/2 mice by reverse transcription-polymerase chain reaction, which indicates that the submandibular gland type is ubiquitous and that its expression is not regulated by the *Gsl5* gene. Results obtained using the long accurate polymerase chain reaction method indicate that the **GNT** gene is approximately 45 kilobases long, and the order of the exons from the 5'-end is exon 1 of the kidney type, exon 1 of the ubiquitous type, exon 2, and exon 3. Exons 2 and 3 are

present in both transcripts, and the translated region is in exon 3. These data suggest that the expression of **GNT** is regulated by an alternative splicing mechanism and also probably by tissue-specific enhancers and that *Gsl5* regulates the expression of **GNT** only in the kidney.

L6 ANSWER 7 OF 16 MEDLINE  
ACCESSION NUMBER: 96228338 MEDLINE  
DOCUMENT NUMBER: 96228338 PubMed ID: 8642259  
TITLE: A peptide recognized by human cytolytic T lymphocytes on HLA-A2 melanomas is encoded by an intron sequence of the N-acetylglucosaminyltransferase V gene.  
AUTHOR: Guilloux Y; Lucas S; Brichard V G; Van Pel A; Viret C; De Plaen E; Brasseur F; Lethe B; Jotereau F; Boon T  
CORPORATE SOURCE: Ludwig Institute for Cancer Research, Brussels Branch and Cellular Genetics Unit, Universite Catholique de Louvain, Belgium.  
SOURCE: JOURNAL OF EXPERIMENTAL MEDICINE, (1996 Mar 1) 183 (3) 1173-83.  
Journal code: 2985109R. ISSN: 0022-1007.  
PUB. COUNTRY: United States  
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
LANGUAGE: English  
FILE SEGMENT: Priority Journals  
OTHER SOURCE: GENBANK-X91652; GENBANK-X91653  
ENTRY MONTH: 199607  
ENTRY DATE: Entered STN: 19960726  
Last Updated on STN: 19970203  
Entered Medline: 19960716

AB A cytolytic T lymphocyte (CTL) **clone** that lyses many HLA-A2 melanomas was derived from a population of tumor-infiltrating lymphocytes of an HLA-A2 melanoma patient. The gene coding for the antigen recognized by this CTL was identified by transfection of a **cDNA** library. It

is the gene which has been reported to code for N-acetylglucosaminyltransferase V (GnT-V). Remarkably the antigenic peptide recognized by the CTL is encoded by a sequence located in an intron. In contrast to the fully spliced GnT-V mRNA, which was found in a wide range of normal and tumoral tissues, the mRNA containing the intron region coding for the antigen was not found at a significant level in normal tissues. This mRNA was observed to be present in about 50% of melanomas. Our results suggest that a promoter located near the end of the relevant intron is activated in melanoma cells, resulting in the production of an mRNA coding for the antigen.

L6 ANSWER 8 OF 16 MEDLINE

ACCESSION NUMBER: 96004617 MEDLINE  
 DOCUMENT NUMBER: 96004617 PubMed ID: 7568011  
 TITLE: Suppression of lung metastasis of B16 mouse melanoma by N-acetylglucosaminyltransferase III gene transfection.  
 AUTHOR: Yoshimura M; Nishikawa A; Ihara Y; Taniguchi S; Taniguchi N  
 CORPORATE SOURCE: Department of Biochemistry, Osaka University Medical School, Japan.  
 SOURCE: PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (1995 Sep 12) 92 (19) 8754-8. Journal code: 7505876. ISSN: 0027-8424.  
 PUB. COUNTRY: United States  
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
 LANGUAGE: English  
 FILE SEGMENT: Priority Journals  
 ENTRY MONTH: 199510  
 ENTRY DATE: Entered STN: 19951227  
 Last Updated on STN: 19980206  
 Entered Medline: 19951023

AB The beta 1-6 structure of N-linked oligosaccharides, formed by **beta-1,6-N-acetylglucosaminyltransferase (GnT-V)**, is associated with metastatic potential. We established a highly metastatic subclone, B16-hm, from low metastatic B16-F1 murine melanoma cells. The gene encoding beta-1,4-N-acetylglucosaminyltransferase (GnT-III) was introduced into the B16-hm cells, and three clones that stably expressed high GnT-III activity were obtained. In these transfectants, the affinity to leukoagglutinating phytohemagglutinin was reduced, whereas the binding to erythroagglutinating phytohemagglutinin was increased, indicating that the level of beta 1-6 structure was decreased due to competition for substrate between intrinsic GnT-V and ectopically expressed GnT-III. Lung metastasis after intravenous injection of the transfectants into syngeneic and nude mice was significantly suppressed, suggesting that the decrease in beta 1-6 structure suppressed metastasis via a mechanism independent of the murine system. These transfectants also displayed decreased invasiveness into Matrigel and inhibited cell attachment to collagen and laminin. Cell growth was not affected. Our results demonstrate a causative role for beta 1-6 branches in invasion and cell attachment in the extravasation stage of metastasis.

L6 ANSWER 9 OF 16 MEDLINE

ACCESSION NUMBER: 95197656 MEDLINE  
 DOCUMENT NUMBER: 95197656 PubMed ID: 7890758  
 TITLE: Transforming growth factor beta up-regulates expression of the N-acetylglucosaminyltransferase V gene in mouse melanoma cells.  
 AUTHOR: Miyoshi E; Nishikawa A; Ihara Y; Saito H; Uozumi N; Hayashi N; Fusamoto H; Kamada T; Taniguchi N  
 CORPORATE SOURCE: Department of Biochemistry, Osaka University Medical School, Japan.

.. SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (1995 Mar 17) 270 (11)  
627-620.  
Journal code: 2985121R. ISSN: 0021-9938.  
PUB. COUNTRY: United States  
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
LANGUAGE: English  
FILE SEGMENT: Priority Journals  
ENTRY MONTH: 199504  
ENTRY DATE: Entered STN: 19950427  
Last Updated on STN: 19970203  
Entered Medline: 19950418

AB **beta-1,6-N-acetylglucosaminyltransferase V (GnT-V) (EC 2.4.1.155)**  
that catalyzes beta-1,6 branching in asparagine-linked oligosaccharides  
is  
activated on viral or oncogenic transformation and is associated with  
tumor metastasis. To study the molecular mechanisms involved in  
regulation  
of expression of the **GnT-V** gene, we cloned **cDNA** and  
genomic DNA for the enzyme (Saito, H., Nishikawa, A., Gu, J., Ihara, Y.,  
Soejima, Y., Sekiya, C., Niikawa, N., and Taniguchi, N. (1994) Biochem.  
Biophys. Res. Commun. 198, 318-327). We found that transforming growth  
factor beta (TGF beta) specifically induced **GnT-V** expression in  
mouse melanoma cells. The activity of **GnT-V** was increased 24 h  
after the addition of TGF beta and remained at high levels up to 72 h.  
Northern blot analysis showed that the mRNA levels of **GnT-V** were  
consistent with the increased activity. To further investigate the nature  
of the induction, mRNA stability and transcriptional activity were  
assayed. The enhancement of the **GnT-V** mRNA expression resulted  
from prolonged mRNA stability, not from increased transcription.  
Consequently, elevated mRNA levels were observed even 72 h after the  
addition of TGF beta. Lectin blot analysis involving leucoagglutinin  
showed newly synthesized beta-1,6 branching structures in the sugar  
chains  
of a protein of approximately 130 kDa at 48 h after TGF beta treatment.  
These results suggested that TGF beta caused changes in the sugar chains  
of proteins in melanoma cells by up-regulating **GnT-V** expression.

L6 ANSWER 10 OF 16 MEDLINE  
ACCESSION NUMBER: 94121649 MEDLINE  
DOCUMENT NUMBER: 94121649 PubMed ID: 8292036  
TITLE: **cDNA** cloning and chromosomal mapping of human  
N-acetylglucosaminyltransferase V+.  
AUTHOR: Saito H; Nishikawa A; Gu J; Ihara Y; Soejima H; Wada Y;  
Sekiya C; Niikawa N; Taniguchi N  
CORPORATE SOURCE: Department of Biochemistry, Osaka University Medical  
School, Japan.  
SOURCE: BIOCHEMICAL AND BIOPHYSICAL RESEARCH COMMUNICATIONS, (1994  
Jan 14) 198 (1) 318-27.  
Journal code: 0372516. ISSN: 0006-291X.  
PUB. COUNTRY: United States  
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
LANGUAGE: English  
FILE SEGMENT: Priority Journals  
OTHER SOURCE: GENBANK-D17716  
ENTRY MONTH: 199402  
ENTRY DATE: Entered STN: 19940312  
Last Updated on STN: 19970203  
Entered Medline: 19940218

AB Human N-acetylglucosaminyltransferase V (**GnT-V**, EC 2.4.1.155)  
**cDNA** was isolated from a human fetal liver **cDNA** library.  
Oligonucleotide primers for polymerase chain reaction were designed  
according to the amino acid sequence of human **GnT-V**. Screening  
for the **cDNA** was carried out by plaque hybridization using PCR  
products of about 500 bp. Human **GnT-V** has 741 amino acids and  
six putative N-glycosylation sites. The homology to rat **GnT-V** is



88% at the nucleotide level and is 97% at the amino acid level, and there is one amino acid insertion. Using the **cDNA clones** probe, five overlapping genomic **clones** have been isolated from a human phagemid DNA library. The **GnT-V** gene has been mapped to chromosome 2q21 using fluorescence in situ hybridization.

L6 ANSWER 11 OF 16 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 2001:761744 CAPLUS

DOCUMENT NUMBER: 136:84455

TITLE: Genetic engineering of CHO cells producing human interferon- $\gamma$ . by transfection of sialyltransferases

AUTHOR(S): Fukuta, Kazuhiro; Yokomatsu, Tomoko; Abe, Reiko; Asanagi, Mineko; Makino, Tadashi

CORPORATE SOURCE: Life Science Laboratory, Mitsui Chemicals, Inc., Chiba, 297-0017, Japan

SOURCE: Glycoconjugate Journal (2001), Volume Date 2000, 17(12), 895-904

CODEN: GLJOEW; ISSN: 0282-0080

PUBLISHER: Kluwer Academic Publishers

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Natural human interferon- $\gamma$ . (hIFN- $\gamma$ .) contains mainly biantennary complex-type sugar chains. We previously remodeled the branch

structures of N-glycans on hIFN- $\gamma$ . in Chinese hamster ovary (CHO) cells by overexpressing UDP-N-acetylglucosamine:  $\alpha$ .1,6-D-mannoside **beta.1,6-N-**

**acetylglucosaminyltransferase (GnT-V)**. Normal CHO cells primarily produced hIFN- $\gamma$ . having biantennary sugar chains, whereas

a CHO **clone**, designated IM4/Vh, transfected with **GnT-V**, primarily produced hIFN- $\gamma$ . having GlcNAc.**beta.1-6** branched triantennary sugar chains when sialylation was incomplete and an increase in poly-N-acetyllactosamine (Gal.**beta.1-4**GlcNAc.**beta.1-3**)<sub>n</sub> was obsd. In the present study, we introduced mouse Gal.**beta.1-3/4**GlcNAc-R  $\alpha$ .2,3-sialyltransferase (ST3Gal IV) and/or rat Gal.**beta.1-4**GlcNAc-R  $\alpha$ .2,6-sialyltransferase (ST6Gal I) **cDNAs** into the IM4/Vh cells to increase the extent of sialylation and to examine the effect of sialyltransferase (ST) type on the linkage of sialic acid. Furthermore, we speculated that sialylation extent might affect the level of poly-N-acetyllactosamine. We isolated four **clones** expressing different levels of  $\alpha$ .2,3-ST and/or  $\alpha$ .2,6-ST. The extent of sialylation of hIFN- $\gamma$ . from the IM4/Vh **clone** was 61.2%, which increased to about 80% in every ST transfectant. The increase occurred regardless of the type of overexpressed ST, and the proportion

of  $\alpha$ .2,3- and  $\alpha$ .2,6-sialic acid corresponded to the activity ratio of  $\alpha$ .2,3-ST to  $\alpha$ .2,6-ST. Furthermore, the proportion of N-glycans contg. poly-N-acetyllactosamine was significantly reduced (less than 10%) in the ST transfectants compared with the parental IM4/Vh **clone** (22.9%). These results indicated that genetic engineering of STs is highly effective for regulating the terminal structures of sugar

chains on recombinant proteins in CHO cells.

REFERENCE COUNT: 42 THERE ARE 42 CITED REFERENCES AVAILABLE FOR THIS

FORMAT RECORD. ALL CITATIONS AVAILABLE IN THE RE

L6 ANSWER 12 OF 16 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 1999:150564 CAPLUS

DOCUMENT NUMBER: 130:322212

TITLE: Control of O-glycan branch formation. Molecular cloning of human **cDNA** encoding a novel **beta.1,6-N-**

**acetylglucosaminyltransferase forming core 2**

and core 4

AUTHOR(S): Schwientek, Tilo; Nomoto, Mitsunaru; Levery, Steven B.; Merkx, Gerard; Van Kessel, Ad Geurts; Bennett, Eric P.; Hollingsworth, Michael A.; Clausen, Henrik  
CORPORATE SOURCE: School of Dentistry, University of Copenhagen, Copenhagen, 2200, Den.

SOURCE: Journal of Biological Chemistry (1999), 274(8), 4504-4512

CODEN: JBCHA3; ISSN: 0021-9258

PUBLISHER: American Society for Biochemistry and Molecular Biology

DOCUMENT TYPE: Journal

LANGUAGE: English

AB A novel human UDP-GlcNAc:Gal/GlcNAc.beta.1-3GalNAc.alpha. .beta.1,6GlcNAc-transferase, designated C2/4GnT, was identified by BLAST anal. of expressed sequence tags. The sequence of C2/4GnT encoded a putative type II transmembrane protein with significant sequence similarity to human C2GnT and IGnT. Expression of the secreted form of C2/4GnT in insect cells showed that the gene product had UDP-N-acetyl-.alpha.-D-glucosamine:acceptor .beta.1, 6-N-acetylglucosaminyltransferase (.beta.1,6GlcNAc-transferase) activity. Anal. of substrate specificity revealed that the enzyme catalyzed O-glycan branch formation of the core

2

and core 4 type. NMR analyses of the product formed with core 3-para-nitrophenyl confirmed the product core 4-para-nitrophenyl. The coding region of C2/4GnT was contained in a single exon and located to chromosome 15q21.3. Northern anal. revealed a restricted expression pattern of C2/4GnT mainly in colon, kidney, pancreas, and small intestine. No expression of C2/4GnT was detected in brain, heart, liver, ovary, placenta, spleen, thymus, and peripheral blood

leukocytes. The expression of core 2 O-glycans has been correlated with cell differentiation processes and cancer. The results confirm the predicted existence of a .beta.1,6GlcNAc-transferase that functions in both core 2 and core 4 O-glycan branch formation. The redundancy in .beta.1,6GlcNAc-transferases capable of forming core 2 O-glycans is important for understanding the mechanisms leading to specific changes in core 2 branching during cell development and malignant transformation.

REFERENCE COUNT: 65 THERE ARE 65 CITED REFERENCES AVAILABLE FOR THIS

RECORD. ALL CITATIONS AVAILABLE IN THE RE

FORMAT

L6 ANSWER 13 OF 16 SCISEARCH COPYRIGHT 2002 ISI (R)

ACCESSION NUMBER: 2000:262310 SCISEARCH

THE GENUINE ARTICLE: 299GV

TITLE: Remodeling of sugar chain structures of human interferon-gamma

AUTHOR: Fukuta K; Abe R; Yokomatsu T; Kono N; Asanagi M; Omae F; Minowa M T; Takeuchi M; Makino T (Reprint)

CORPORATE SOURCE: MITSUBISHI CHEM INC, LIFE SCI LAB, 1144 TOGO, CHIBA 2970017, JAPAN (Reprint); MITSUBISHI CHEM INC, LIFE SCI LAB, CHIBA 2970017, JAPAN; KIRIN BREWERY CO LTD, CENT

LABS

KEY TECHNOL, KANAZAWA KU, YOKOHAMA, KANAGAWA 236000,

JAPAN

COUNTRY OF AUTHOR: JAPAN

SOURCE: GLYCOBIOLOGY, (APR 2000) Vol. 10, No. 4, pp. 421-430. Publisher: OXFORD UNIV PRESS, GREAT CLARENDON ST, OXFORD OX2 6DP, ENGLAND. ISSN: 0959-6658.

DOCUMENT TYPE: Article; Journal

FILE SEGMENT: LIFE

LANGUAGE: English

AB Natural human Interferon (IFN)-gamma has mainly biantennary complex-type sugar chains and scarcely has multiantennary structures. We attempted to remodel the sugar chain structures using IFN-gamma as a model glycoprotein. To obtain the branching glycoforms of IFN-gamma, we introduced the genes for **GnT-IV** (UDP-N-acetylglucosamine:alpha-1,3-D-mannoside beta-1,4-N-acetylglucosaminyltransferase) and/or **GnT-V** (UDP-N-acetylglucosamine :alpha-1,6-D-mannoside **beta-1,6-N-acetylglucosaminyltransferase**) into Chinese hamster ovary (CHO) cells producing human IFN-gamma. The parental CHO cells produced IFN-gamma with biantennary sugar chains mainly. When the **GnT-IV** activity was increased, triantennary sugar chains with a branch produced by **GnT-IV** increased up to 66.9% of the total sugar chains. When the **GnT-V** activity was increased, triantennary sugar chains with a corresponding branch increased up to 55.7% of the total sugar chains. When the **GnT-IV** and -V activities were increased at a time, tetraantennary sugar chains increased up to 56.2% of the total sugar chains. The proportion of these multiantennary sugar chains corresponded to the intracellular activities of **GnT-IV** and -V. What is more, lectin blot and flow cytometric analysis indicated that the multi-branch structure of the sugar chains was increased not only on IFN-gamma, one of the secretory glycoproteins, but also on almost CHO cellular proteins by introducing either or both of the **GnT** genes. The results suggest that the branching structure of sugar chains of glycoproteins could be controlled by cellular **GnT-IV** and **GnT-V** activities. This technology can produce glycoforms out of natural occurrence, which should enlarge the potency of glycoprotein therapeutics.

L6 ANSWER 14 OF 16 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.

ACCESSION NUMBER: 2000:309153 BIOSIS

DOCUMENT NUMBER: PREV200000309153

TITLE: Expression of a specific glycosyltransferase enzyme regulates T cell death mediated by galectin-1.

AUTHOR(S): Galvan, Marisa; Tsuboi, Shigeru; Fukuda, Minoru; Baum, Linda G. (1)

CORPORATE SOURCE: (1) Dept. of Pathology and Laboratory Medicine, UCLA School

of Medicine, 10833 Le Conte Ave., Los Angeles, CA, 90095 USA

SOURCE: Journal of Biological Chemistry, (June 2, 2000) Vol. 275, No. 22, pp. 16730-16737. print. ISSN: 0021-9258.

DOCUMENT TYPE: Article

LANGUAGE: English

SUMMARY LANGUAGE: English

AB Galectin-1 induces apoptosis of immature thymocytes and activated T cells,

suggesting that galectin-1 regulates cell death in the thymus during selection and in the periphery following an immune response. Although it is known that galectin-1 recognizes lactosamine (Gal-GlcNAc) as a minimal ligand, this disaccharide is ubiquitously expressed on a variety of cell surface glycoproteins. Thus, susceptibility to galectin-1 may be regulated

by the presentation of lactosamine on specific oligosaccharide structures created by specific glycosyltransferase enzymes. The core 2 **beta-1,6-N-acetylglucosaminyltransferase**

(core 2 **GnT**) creates a branched structure on O-glycans that can be elongated to present multiple lactosamine sequences. In the thymus,

the core 2 **GnT** is expressed in galectin-1-sensitive thymocyte

subsets. In the periphery, an oligosaccharide epitope created by the core 2 **GnT** is expressed on galectin-1-sensitive activated T-cells. In this report, we demonstrate that expression of the core 2 **GnT** was necessary and sufficient for galectin-1-induced death of murine T cell lines. In addition, over-expression of the core 2 **GnT** in mice increased the susceptibility of double positive thymocytes to galectin-1. These data demonstrate that expression of a specific glycosyltransferase can control susceptibility to galectin-1, suggesting that developmentally regulated glycosyltransferase expression may be a mechanism to modulate cell death during T cell development and function.

L6 ANSWER 15 OF 16 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.

ACCESSION NUMBER: 2001335139 EMBASE

TITLE: Genetic engineering of CHO cells producing human interferon- $\gamma$  by transfection of sialyltransferases.

AUTHOR: Fukuta K.; Yokomatsu T.; Abe R.; Asanagi M.; Makino T.

CORPORATE SOURCE: K. Fukuta, Life Science Laboratory, Mitsui Chemicals, Inc.,

1144 Togo, Mobara, Chiba 297-0017, Japan.

kazuhiro.fukuta@mitsui-chem.co.jp

SOURCE: Glycoconjugate Journal, (2000) 17/12 (895-904).

Refs: 42

ISSN: 0282-0080 CODEN: GLJOEW

COUNTRY: Netherlands

DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 027 Biophysics, Bioengineering and Medical Instrumentation

029 Clinical Biochemistry

LANGUAGE: English

SUMMARY LANGUAGE: English

AB Natural human interferon- $\gamma$ . (h1FN- $\gamma$ ) contains mainly biantennary complex-type sugar chains. We previously remodeled the branch structures of N-glycans on h1FN- $\gamma$  in Chinese hamster ovary (CHO) cells by overexpressing UDP-N-acetylglucosamine:  $\alpha$ -1,6-D-mannoside  $\beta$ -1,6-N-acetylglucosaminyltransferase (**GnT-V**). Normal CHO cells primarily produced h1FN- $\gamma$  having biantennary sugar chains, whereas

a CHO clone, designated IM4/Vh, transfected with **GnT-V**, primarily produced h1FN- $\gamma$  having GlcNAc $\beta$ -1-6 branched triantennary sugar chains when sialylation was incomplete and an increase in poly-N-acetyllactosamine (Gal $\beta$ -1-4GlcNAc $\beta$ -1-3) $_n$  was observed. In the present study, we introduced mouse Gal $\beta$ -1-3/4GlcNAc-R  $\alpha$ -2,3-sialyltransferase (ST3Gal IV) and/or rat Gal $\beta$ -1-4GlcNAc-R  $\alpha$ -2,6-sialyltransferase (ST6Gal I) cDNAs into the IM4/Vh cells to increase the extent of sialylation and to examine the effect of sialyltransferase (ST) type on the linkage of sialic acid. Furthermore,

we speculated that sialylation extent might affect the level of poly-N-acetyllactosamine. We isolated four clones expressing different levels of  $\alpha$ -2,3-ST and/or  $\alpha$ -2,6-ST. The extent of sialylation of h1FN- $\gamma$  from the IM4/Vh clone was 61.2%, which increased to about 80% in every ST transfectant. The increase occurred regardless of the type of overexpressed ST, and the proportion

of  $\alpha$ -2,3- and  $\alpha$ -2,6-sialic acid corresponded to the activity ratio of  $\alpha$ -2,3-ST to  $\alpha$ -2,6-ST. Furthermore, the proportion of N-glycans containing poly-N-acetyllactosamine was significantly reduced (less than 10%) in the ST transfectants compared with the parental IM4/Vh clone (22.9%). These results indicated that genetic engineering of STs is highly effective for regulating the terminal structures of sugar chains on recombinant proteins in CHO cells.

L6 ANSWER 16 OF 16 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.

ACCESSION NUMBER: 2001124619 EMBASE

TITLE: Relationship between metastasis-associated phenotypes and N-glycan structure of surface glycoproteins in human hepatocarcinoma cells.  
AUTHOR: Guo H.-B.; Zhang Y.; Chen H.-L.  
CORPORATE SOURCE: H.-L. Chen, Key Lab. of Glycoconjugate Res., Ministry of Health, Department of Biochemistry, Shanghai 200032, China.

hlchen@shmu.cdu.cn  
SOURCE: Journal of Cancer Research and Clinical Oncology, Supplement, (2001) 127/4 (231-236).

Refs: 24

ISSN: 0943-9382 CODEN: JCCSED

COUNTRY: Germany

DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 016 Cancer

LANGUAGE: English

SUMMARY LANGUAGE: English

AB Purpose: To study the relation of N-glycan structure on cell surface glycoproteins to the metastatic phenotypes. Methods: Two human hepatocarcinoma 7721 cell lines transfected with sense or antisense cDNA of **GnT-V**, named **GnT-V/7721** and **GnT-V-AS/7721**, respectively, were adopted, because **GnT-V** can change the antennary number and the content of the .beta.1,6 Glc-NAc branch in N-glycans. The effects of over- and under-expression of **GnT-V** on the metastasis-associated phenotype of the transfected cells were investigated and compared with the cells mock-transfected with the plasmid vector. Results: In **GnT-V/7721** cells, **GnT-V** activity was increased by 92% compared with the mock cells.

HRP-labeled

lectin staining of transfected cells showed elevated intensity with HRP-L-PHA and reduced intensity with HRP-ConA, suggesting the increased antennary number and content of the .beta.1,6 GlcNAc branch in N-

glycans.

Analysis of the N-glycan structure of [(3)H]-labeled glycopeptides prepared from cell-surface [(3)H] glycoproteins using DSA-affinity chromatography also revealed the above change of the N-glycan structure

in

a more quantitative manner. **GnT-V/7721** cells showed a suppressed cell attachment to fibronectin (Fn) or laminin (Ln), and increased cell migration and invasion through matrigel. In contrast, **GnT-V-AS/7721** cells showed reduction of both **GnT-V** activity and content of the .beta.1,6 branch in N-linked glycans, elevation of cell attachment to Fn or Ln, and decline of cell migration and invasion

through

matrigel. These changes were just the opposite to those in **GnT-V/7721** cells. Conclusions: The alteration of N-glycan structure in surface glycoproteins resulting from the activity change of **GnT-V** contributes to the alterations in metastasis-associated phenotypes.

The

product of **GnT-V**, the .beta.1,6 GlcNAc branch in N-linked glycans, is a structural factor of adhesion inhibition and invasion promotion. **GnT-V** is, therefore, closely related to cancer metastasis and its over-expression is an important molecular mechanism of metastasis.